ABC of palliative care - Emergencies

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Emergencies

Stephen Falk, Marie Fallon

The concept of rapid assessment, evaluation, and management of symptoms due to malignancy is generally accepted. Inherent in this concept is rapid reversal of what is reversible. Some acute events in malignancy have to be treated as an emergency if a favourable outcome is to be achieved. As in any emergency, the assessment must be as prompt and complete as possible. In patients with advanced malignancy, factors to consider include

- The nature of the emergency
- The general physical condition of the patient
- Disease status and likely prognosis
- Concomitant pathologies
- Symptomatology
- The likely effectiveness and toxicity of available treatments
- Patients' and carers' wishes.

While unnecessary hospital admission may cause distress for the patient and carers, missed emergency treatment of reversible symptomatology can be disastrous.

Major emergencies in palliative care

- Hypercalcaemia
- Superior venal caval obstruction
- Spinal cord compression
- Bone fractures

Other emergencies, such as haemorrhage and acute anxiety and depression, are discussed elsewhere in this series

Questions to ask when considering management of emergencies in patients with advanced disease

- What is the problem?
- Can it be reversed?
- What effect will reversal of the symptom have on patient's overall condition?
- What is your medical judgment?
- What does the patient want?
- What do the carers want?
- Could active treatment maintain or improve this patient's quality of life?

Hypercalcaemia

Hypercalcaemia is the commonest life threatening metabolic disorder encountered in patients with cancer. The incidence varies with the underlying malignancy, being most common in multiple myeloma and breast cancer (40-50%), less so in non-small cell lung cancer, and rare in small cell lung cancer and colorectal cancer.

It is important to remember non-malignant causes of hypercalcaemia—particularly primary hyperparathyroidism, which is prevalent in the general population.

The pathology of hypercalcaemia is mediated by factors such as parathyroid related protein, prostaglandins, and local interaction by cytokines such as interleukin 1 and tumour necrosis factor. Bone metastases are commonly but not invariably present.

Presenting features of hypercalcaemia

Mild symptoms

- Nausea
- Anorexia and vomiting
- Constipation
- Thirst and polyuria

Severe symptoms and signs

- Gross dehydration
- Drowsiness
- Confusion and coma
- Abnormal neurology
- Cardiac arrhythmias

Management

Mild hypercalcaemia (corrected serum calcium concentration ≤3.00 mmol/l) is usually asymptomatic, and treatment is required only if a patient has symptoms. For more severe hypercalcaemia, however, treatment can markedly improve symptoms even when a patient has advanced disease and limited life expectancy to make the end stages less traumatic for patient and carers.

Treatment with bisphosphonate normalises the serum calcium concentration in 80% of patients within a week. Treatment with calcitonin or mithramycin is now largely obsolete. Corticosteroids are probably useful only when the underlying tumour is responsive to this cytostatic agent—such as myeloma, lymphoma, and some carcinomas of the breast.

Some symptoms, particularly confusion, may be slow to improve after treatment despite normalisation of the serum calcium. Always consider treating the underlying malignancy to prevent recurrence of symptoms, since the median duration of normocalcaemia after bisphosphonate infusion is only three weeks. However, if effective systemic therapy has been exhausted, or is deemed inappropriate, oral bisphosphonates (such as clodronate 800 mg twice daily) or parenteral infusions (every three to four weeks) should be considered.

Management of hypercalcaemia

1. Check serum concentration of urea, electrolytes, albumin, and calcium

2. Calculate corrected calcium concentration

- Corrected Ca = measured Ca + (40 – albumin) × 0.02 mmol/l
- Corrected calcium value is used for treatment decisions

3. Rehydrate with intravenous fluid (0.9% saline)

- Amount and rate depends on clinical and cardiovascular status and concentrations of urea and electrolytes

4. After minimum of 2 l of intravenous fluids give bisphosphonate infusion

- Disodium pamidronate (60 mg if Ca < 3.5 mmol/l, 90 mg if Ca ≥3.5 mmol/l) over 2 hours *or*
- Sodium clodronate 1500 mg over 4 hours
- Both given in 0.5 litre 0.9% saline

5. Measure concentrations of urea and electrolytes at daily intervals and give intravenous fluids as necessary

- Normalisation of serum calcium takes 3-5 days
- Do not measure serum calcium for at least 48 hours after rehydration as it may rise transiently immediately after treatment

6. Prevent recurrence of symptoms

- Treat underlying malignancy if possible *or*
- Consider maintenance treatment with bisphosphonates and monitor serum calcium at 3 week intervals *or*
- Monitor serum calcium at 3 week intervals, or less if patient symptomatic, and repeat bisphosphonate infusion as appropriate

Maintenance intravenous bisphosphonates may be administered at a day centre or outpatient department. Oral preparations have the disadvantages of being poorly absorbed and have to be taken at least one hour before or after food. The evidence for intravenous or oral bisphosphonates is equal, and choice depends on the individual.

Superior venal caval obstruction

This may arise from occlusion by extrinsic pressure, intraluminal thrombosis, or direct invasion of the vessel wall. Most cases are due to tumour within the mediastinum, of which up to 75% will be primary bronchial carcinomas. About 3% of patients with carcinoma of the bronchus and 8% of those with lymphoma will develop superior venal caval obstruction.

Management

Conventionally, superior venal caval obstruction has been regarded as an oncological emergency requiring immediate treatment. If it is the first presentation of malignancy, treatment will be tempered by the need to obtain an accurate histological diagnosis in order to tailor treatment for potentially curable diseases, such as lymphomas or germ cell tumours, and for diseases such as small cell lung cancer that are better treated with chemotherapy at presentation.

In advanced disease patients need relief of acute symptoms—of which dyspnoea and a sensation of drowning can be most frightening—and high dose corticosteroids and radiotherapy should be considered. In non-small cell lung cancer palliative radiotherapy gives symptomatic improvement in 70% of patients, with a median duration of palliation of three months. Up to 17% of patients may survive for a year. If radiotherapy is contraindicated or being awaited corticosteroids alone (dexamethasone 16 mg/day) may give relief. In those for whom further radiotherapy is not indicated, stenting (with or without thrombolysis) of the superior vena cava should be considered.

Urgent initiation of pharmacological, practical, and psychological management of dyspnoea is paramount and usually includes opioids, with or without benzodiazepines. Opioid doses are usually small—such as 5 mg morphine every 4 hours. It is important to review all corticosteroid prescriptions in view of their potential adverse effects. We recommend stopping corticosteroids after five days if no benefit is obtained, and a gradual reduction in dose for those who have responded.

Spinal cord compression

Presentation of spinal cord compression can be very subtle in the early stages. Any patient with back pain and subtle neurological symptoms or signs should have radiological investigations, with magnetic resonance imaging when possible

This occurs in up to 5% of cancer patients. The main problem in clinical practice is failure of recognition. It is not uncommon for patients' weak legs to be attributed to general debility, and urinary and bowel symptoms to be attributed to medication. Neurological symptoms and signs can vary from subtle to gross, from upper motor neurone to lower motor neurone, and from minor sensory changes to clearly demarcated sensory loss.

Prompt treatment is essential if function is to be maintained: neurological status at the start of treatment is the most important factor influencing outcome. If treatment is started within 24-48 hours of onset of symptoms neurological damage may be reversible.

Aetiology of superior venal caval obstruction

- Carcinoma of the bronchus 65-80%
- Lymphoma 2-10%
- Other cancers 3-13%
- Benign causes now rare
  - Benign goitre
  - Aortic aneurysm (syphilis)
  - Thrombotic syndromes
  - Idiopathic sclerosing mediastinitis
- Unknown or undiagnosed 5%

Patient with superior venal caval obstruction showing typical signs (reproduced with patient's permission)

Clinical features of superior venal caval obstruction

Symptoms

- Tracheal oedema and shortness of breath
- Cerebral oedema with headache worse on stooping
- Visual changes
- Dizziness and syncope
- Swelling of face, particularly periorbital oedema
- Neck swelling
- Oedema of arms and hands

Clinical signs

- Rapid breathing
- Periorbital oedema
- Suffused injected conjunctivae
- Cyanosis
- Non-pulsatile distension of neck veins
- Dilated collateral superficial veins of upper chest
- Oedema of hands and arms

Spinal cord compression can arise from intradural metastasis but is more commonly extradural in origin. In 85% of cases cord damage arises from extension of a vertebral body metastasis into the epidural space, but other mechanisms of damage include vertebral collapse, direct spread of tumour through the intervertebral foramen (usually in lymphoma or testicular tumour), and interruption of the vascular supply.

The frequency with which a spinal level is affected reflects the number and volume of vertebral bodies in each segment—about 10% of compressions are cervical, 70% thoracic, and 20% lumbosacral. It is important to remember that more than one site of compression may occur, and this is increasingly recognised with improved imaging techniques.

The earliest symptom of spinal cord compression is back pain, sometimes with symptoms of root irritation, causing a girdle-like pain, often described as a "band," that tends to be worse on coughing or straining. Most patients have pain for weeks or months before they start to detect weakness. Initially, stiffness rather than weakness may be a feature, and tingling and numbness usually starts in both feet and ascends the legs. In contrast to pain, the start of myelopathy is usually rapid. Urinary symptoms such as hesitancy or incontinence and perianal numbness are late features. Increasing compression of the spinal cord is often marked by improvement or resolution of the back pain but can be associated with worsening of pain.

Examination may reveal a demarcated area of sensory loss and brisk or absent reflexes, which may help to localise the lesion. In patients unfit to undergo more detailed investigations, plain radiology can reveal erosion of the pedicles, vertebral collapse, and, occasionally, a large paravertebral mass. These may help in the application of palliative radiotherapy. In contrast to myelography with localised computed tomographic *x* rays for soft tissue detail, magnetic resonance imaging is now considered the investigation of choice: it is non-invasive and shows the whole spine, enabling detection of multiple areas of compression.

Magnetic resonance image showing patient with spinal cord compression at two different sites (arrows)

Management

Decisions on investigations performed and treatment given will depend on the patient's wishes and the stage of the disease. Only in exceptional circumstances will corticosteroids not form part of the treatment plan

After palliative radiotherapy, 70% of patients who were ambulatory at the start of treatment retain their ability to walk and 35% of paraparetic patients regain their ability to walk, while only 5% of completely paraplegic patients do so. These figures underline the importance of early diagnosis, since 75% of patients have substantial weakness at presentation to oncology units.

Retrospective analysis has not shown an advantage for patients managed by laminectomy and radiotherapy over radiotherapy alone. Surgical decompression is therefore now performed less routinely and is usually reserved for cases when
- A tissue diagnosis is required (if biopsy guided by computed tomography is not possible)
- Deterioration occurs during radiotherapy
- There is bone destruction causing spinal cord compression.

For a small number of fit patients with disease anterior to the spinal canal, excellent results have been reported for an anterior approach for surgical decompression and vertebral stabilisation—80% of the patients became ambulant. For relief of the mechanical problems due to bone collapse, laminectomy decompression has to be accompanied by spinal stabilisation. Such surgery is difficult and not always appropriate.

Management of spinal cord compression

Main points
- Except for unusual circumstances give oral dexamethasone 16 mg/day
- Urgent treatment, definitely within 24 hours of start of symptoms
- Interdisciplinary approach involving oncologists, neurosurgeons, radiologists, nurses, physiotherapists, occupational therapists

Treatment options
- Continue with dexamethasone 16 mg/day *plus*
- Radiation only
  - For most situations
  - Radiosensitive tumour without spinal instability
- Surgery and radiation
  - Spinal instability, such as fracture or compression by bone
  - No tissue diagnosis (when needle biopsy guided by computed tomography not possible)
- Surgery only
  - Relapse at previously irradiated area
  - Progression during radiotherapy
- Chemotherapy
  - Paediatric tumours responsive to chemotherapy
  - Adjuvant treatment for adult tumours responsive to chemotherapy
  - Relapse of previously irradiated tumour responsive to chemotherapy
- Corticosteroids alone
  - Final stages of terminal illness and patient either too unwell to have radiotherapy or unlikely to live long enough to have any benefits

Bone fracture

Bone metastases are a common feature of advanced cancer. Bone fracture may also be due to osteoporosis or trauma. Fractures can present in a variety of forms, including as an acute confusional state.

Management

If fracture of a long bone seems likely, as judged by the presence of cortical thinning, prophylactic internal fixation should be considered. Once a fracture has occurred the available options include external or internal fixation—their relative merits are determined by the site of the fracture and the general condition of the patient.

Radiotherapy is usually given in an attempt to enhance healing and to prevent further progression of the bony metastasis and subsequent loosening of any fixation. Evidence exists that, when combined with oncolytic therapy in breast cancer and multiple myeloma, oral bisphosphonates can reduce skeletal morbidity (hypercalcaemia, vertebral fracture, and need for palliative radiotherapy).

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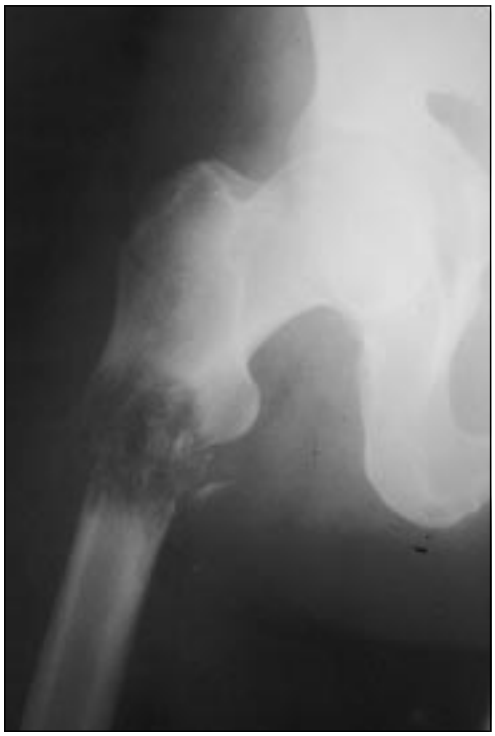
The ABC of palliative care is edited by Marie Fallon and Bill O'Neill, science and research adviser, British Medical Association, BMA House, London. It will be published as a book in June 1998.



Radiograph showing pathological fracture of the femur

Lesson of the week

Long term sequelae of missed tendon injuries at the ankle

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Patients with ankle wounds due to penetrating trauma should be investigated for tendon injury

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BMJ 1997;315:1528–9

Lacerations of the hand and wrist may affect underlying tendons.[1] Although lacerations of the ankle are uncommon, injury to underlying structures is still possible. We report on four patients who presented an average of 23 years after injury to the ankle with the consequences of undetected tendon injuries.

Case reports

Case 1–A 43 year old man presented with a painful left flat foot. At 6 years of age he had sustained a laceration with glass below his left medial malleolus. At the time his family did not seek medical attention. Subsequently the inside sole of his left shoe was seen to be wearing out. At 13 years of age he was referred to an orthopaedic clinic and underwent an unsuccessful tendon repair. Inspection showed a 1 cm transverse laceration below his medial malleolus and a fixed pes planus deformity. His hindfoot was in 10° of fixed valgus (figure) and the ankle in 5° of fixed equinus. The patient's ankle-hindfoot and midfoot scores were 22 and 16 respectively with the scoring system of the American Orthopedic Foot and Ankle Society for grading the functional status of the foot and ankle. The maximum score is 100 and lower scores reflect increasing severity of symptoms and disability.[2] An *x* ray film showed degenerative changes of the ankle and hindfoot joints, and ultrasonography of the ankle showed a discontinuity of the tibialis posterior tendon.

Case 2–A 35 year old woman presented with a painful flat left foot. At 8 years of age she had stood on a broken bottle, causing a laceration below her left medial malleolus. The wound was sutured but not examined in detail. A year later her mother reported that her daughter had a persistent limp and her foot was externally rotated. The patient attended an orthopaedic clinic and subsequently underwent an unsuccessful tendon repair. Clinical examination showed a small scar below the medial malleolus and a fixed pes planus deformity. The hindfoot was in 15° of fixed valgus and 40° of external rotation, and the ankle was in 5° of fixed equinus. The patient was unable to go up on to the toes of her left foot and had a noticeable antalgic gait. The ankle-hindfoot and midfoot scores were 10 and 11. Ultrasonography of the ankle showed a large discontinuity of the tibialis posterior tendon.

Case 3–A 29 year old housewife presented with pain in the hindfoot and midfoot. At 8 years of age she had sustained a laceration with glass to her right ankle below the lateral malleolus. The wound was sutured at a casualty department but not examined in detail. Subsequently she could only wear flat shoes. Clinical examination revealed a small scar behind her right lateral malleolus and callous formation over the lateral border of the sole of the foot. There was a fixed 5° varus deformity of the hindfoot and ankle movement was from 20° of dorsiflexion to 20° of plantar flexion. The patient's ankle-hindfoot and midfoot scores were 52 and 43 respectively. Ultrasonography of the ankle showed a large discontinuity of the peroneus longus tendon.

Case 4–A 15 year old schoolgirl presented with persistent foot pain. At 7 years of age she had sustained a laceration with glass below her right lateral malleolus. The wound had been sutured by her doctor but not examined in detail. The parents reported a persistent inversion of their daughter's right foot, and at 11 years of age she was referred to an orthopaedic clinic. She subsequently underwent two unsuccessful tendon reconstructions. Examination showed a scar below the right medial malleolus and noticeable wasting of the right calf muscle. There was a 10° fixed varus deformity of the hindfoot, and movement at the ankle was from 10° of dorsiflexion to 5° of plantar flexion. The patient's ankle-hindfoot and midfoot scores were 52 and 47 respectively. Ultrasonography of the ankle showed a discontinuity of the peroneus brevis tendon.

Discussion

All four injuries were caused by broken glass. At the time of the injury two of the patients had attended an accident and emergency department, one patient had seen a doctor, and one patient had not sought medical attention. None of the patients had had a detailed examination of the wound. Three of the patients were referred to orthopaedic surgeons for further assessment several years after the injury. In all four patients discontinuity of the tendons was confirmed by ultrasonography. Ultrasonography is a useful diagnostic tool as it is both sensitive and specific in detecting soft tissue disease such as tendon injury. In cases of injury to shoulder tendons ultrasonography has a specificity of 91% to 100% and a sensitivity of 83% to 100%[3]; it detects tendon attenuation as an area of low echogenicity. In the case of our patients ultrasonography was used to delineate the tendon defect and to measure the distance between the proximal and distal ends of the tendon.

In adults degenerate rupture of the tibialis posterior tendon is a recognised cause of painful flat foot[4] while in younger age groups it may be caused by subtalar irritability possibly associated with a tarsal coalition. Laceration of the tibialis posterior tendon is a recognised cause of valgus foot and flat foot in childhood,[5 6] whereas peroneal tendon rupture occurs less commonly; there are only two reports of tendon rupture in patients under 25 years of age. Tendon rupture is usually caused by attrition, as was shown in 14 out of 124 post mortem examinations.[7] The tibialis posterior tendon is tightly held by a sheath around the medial malleolus, and the peroneal tendons are tightly held by a sheath below and behind the lateral malleolus–all three are very close to the skin and therefore vulnerable to lacerations. A penetrating trauma causing laceration of the ankle tendon is fairly uncommon and may be difficult to detect clinically. A patient presenting with an injury to the peroneal tendon may have difficulty everting the foot, while a patient with an injury to the tibialis posterior tendon may have difficulty in performing a single toe raise. Injuries to the peroneal and tibialis posterior tendons are painful, and clinical examination might not reflect any distinct tendon abnormality as foot and ankle movement will be limited by pain. The ankle-hindfoot and midfoot scoring systems provide an objective measure of functional disability at the ankle joint and foot. The scoring system provides information on foot pain, walking ability, the need for orthoses, gait, and movement at each joint. The low scores in our four patients reflect poor disability. Ultimately these patients may require a triple arthrodesis of the hindfoot–resulting in a completely stiff albeit painless hindfoot.

Left hindfoot of patient in case 1 in 10° of valgus. Too many toes sign in evidence

As missed ankle tendon lacerations may lead to disabling and painful fixed foot deformities, patients with ankle wounds due to penetrating trauma should be suspected of tendon injury. These patients should have their wound examined in detail; any tendon injury should be repaired using a modified Kessler suture and the patient immobilised in a cast for four to six weeks until the tendon has healed.

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(Accepted 26 August 1997)

Endpiece

Unavoidable

Accident: An inevitable occurrence due to the action of immutable natural laws.

Ambrose Bierce, *The Cynic's Word Book* (1906), subsequently titled *The Devil's Dictionary*